A Dissertation on

# Effect of Right Ventricular Infarction on the immediate prognosis of Inferior Wall Myocardial Infarction.

**submitted to**
**THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY**
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# Aim of study

1. **To study the incidence of Right Ventricular Infarction (RVI) in**

**patients with Inferior wall myocardial infarction (IWMI), the risk factors, clinical profile and other parameters.**

2. **To find out the influence of RVI on the immediate prognosis of IWMI.**

# Introduction

**RVI is typically associated with [1,2] Inferior wall myocardial infarction involving the interventricular septum and with occlusion of the [3] proximal right coronary artery. Isolated RVI seen in 3-5% of autopsy proven cases of myocardial infarction.**

**Inferior wall myocardial infarction typically involve left ventricular inferoposterior wall, septum and posterior right ventricular free wall [4].**

**Diagnosing RVI is important because they may have important hemodynamic consequences [5] and high incidence of advanced atrioventricular nodal block [6].**

**Studies have identified right ventricular involvement as a major**

**negative prognostic indicator and an independent risk factor for death in patients with inferior wall myocardial infarction , and this risk increases with increasing age [7,8] .**

# Review of Literature

## Historical perspective

**In 1930, Sanders [9] reported the first clinical description of RVI. Several recent studies [10,11] (Cohn etal 1974, Rigo et al 1975 Sharpe et al 1978, Tobinick et al, 1978) have focused attention on the role of right ventricular infarction when cardiogenic shock is associated with IWMI.**

**In 1974, Cohn and colleagues [5] called attention to RVI as a unique clinical and hemodynamic syndrome, characterized in its extreme form by shock , distended neck veins and clear lung fields.**

**In 1970's Erhadt et al [12] showed that a true right ventricular lead (lead $V_4R$) was of value in diagnosis of RVI in patients with acute IWMI.**

**Zeymer et al [13] found that the presence of ST segment elevation of atleast 0.1mv in lead $V_4R$ was associated with increased mortality when accompanied by large ST segment deviations in inferior and left precordial leads.**

**In 1993 Zehander et al [14] showed that RV involvement had serious prognostic consequences in patients admitted to hospital with acute IPMI.**

# Coronary circulation and the RV

**The RCA supplies right ventricular free wall, except for anterior margin, which is supplied by the left anterior descending artery. The posterior descending branch of RCA supplies the posterior wall of right ventricle, as well as posterior surface of left ventricle. Right marginal artery of RCA supplies lateral wall of right ventricle. RCA also supply posteroinferior one third of interventricular septum, the right atrium, part**

**of left atrium and conducting system as far as right and left crura.**

**The LCA divides into left anterior descending artery which supplies the free wall of LV adjacent to interventricular septum and the left circumflex artery which supplies most of left atrium and lateral wall of left ventricle.**

**The SA node (in 65%) and AV node (in 80%) are supplied by RCA. In the rest, they are supplied by left circumflex artery. Variations in blood supply of diaphragmatic aspect of ventricle by posterior interventricular artery determines dominance. In right dominant circulation (85%) posterior interventricular artery , a branch of RCA supplies and in left dominant circulation (5%) it arises from left circumflex artery.**

Right coronary artery with corresponding regions of perfusion and ECG findings for underperfusion..

| Arterial segment | **Arterial branch** | **Perfusion region** | **ECG effects of ischaemia** |
|---|---|---|---|

| | | | |
|---|---|---|---|
| 1) Proximal segment | **SA nodal branch** | **SA node** | **Atrial infarct pattern / AF** |
| | **Right atrial free wall** | **Atrial free wall** | |
| 2) Middle segment | **Lateral RV branch** | **Lateral RV free wall** | **ST segment elevation later abnormal Q waves in $V_3R$ through $V_5R$** |
| | **Marginal RV branch** | **Inferior (posterior) RV free wall** | |
| 3) Distal segment | **AV nodal branch** | **AV node** | **AV block** |
| 4) Posterior descending segment | **Posterior lateral LV branches** | **Posterior left ventricle** | **ST segment elevation later abnormal Q waves in $L_2L_3$ & aVF** |
| | **Posterior descending artery** | **Inferior septum, Inferior left ventricular free wall** | |

**The anterior free wall of RV has a dual blood supply, from conus branch of RCA and the moderator band artery [15], which courses from left anterior descending artery. Moderator band artery provides major source of flow to anterior papillary muscle of right ventricle [16]. Right ventricular infarction usually involves posterior septum and posterior wall rather than right ventricular free wall.**

**Collateral flow to the right ventricle especially through moderator band artery, protects against massive infarction in the presence of proximal right coronary artery occlusion [17-19].**

Factors protecting right ventricle from ischaemic injury.

1. **Lower oxygen requirement of right ventricle by virtue of its smaller muscle mass and lower intracavitary pressure.**
2. **Greater systolic coronary artery blood flow in right ventricle. Extensive potential collateralization of right ventricle including that provided by the left coronary arterial system [20-21].**
3. **Thinner right ventricular wall can derive more nutrition from blood within the right ventricular cavity [22].**

**RV infarction may occur in anterior infarction as its anterior wall may receive some blood from left anterior descending artery [23].**

Isolated right ventricular infarction is rare [23]. But can occur in the following conditions.

1. **Isolated occlusion of right ventricular branches.**
2. **Occlusion of non dominant right coronary artery.**

3. **Occlusion of dominant right coronary artery with good collateral flow to the posterior descending artery.**

# Pathophysiology

**Acute right ventricular distension within the restraining pericardium may flatten the interventricular septum towards the left ventricle during diastole, restricting left ventricular end diastolic pressure [24]. Thus right ventricular infarction may compromise cardiac output, culminating in cardiogenic shock. In animal models with the pericardium removed, it is difficult to induce hypotension with RVI. When pericardium is left intact [25,26] however RVI is associated with the full syndrome, as originally described by Cohn and coworkers[11]**

**Higher incidence of advanced heart block may be explained by the fact that the right ventricle and AV node share a common origin of blood supply.**

**Sympathetic activation secondary to the low output state is proarrythmogenic, and it is possible that infarction of RV may itself be more arrythmogenic than infarction of LV.**

## Effects of right ventricular ischaemia

**Systolic RV dysfunction ⇨ ↓ RVCO ⇨ ↓ LVCO**

**RV dilatation ⇨ ↑RV volume ⇨ septal shift**

**Diastolic RV dysfunction → ↓ compliance →**

**↑RVDP → ↑ Septal shift → ↓ LVDP → ↓ LVCO**

**↑ RAP → ↑ Septal shift → ↓LAP → ↓ LVCO**

**Except in rare cases of isolated RVI [27,28] , LV infarction accompanies RVI. So, decrease in LV function further decreases cardiac output [29].**

**RVCO - Right Ventricular Cardiac Output**

**LVCO - Left Ventricular Cardiac Output**

**RVDP - Right Ventricular Diastolic Pressure**

**LVDP - Left Ventricular Diastolic Pressure**

**RAP - Right Atrial Pressure**

**LAP - Left Atrial Pressure**

# Clinical Presentation

**Hemodynamically significant RVI typically presents with hypotension, jugular vein distension, and occasionally shock, all in the presence of clear lung fields [30].**

**Prominent 'a' wave in JVP and positive kussmaul's sign may be noted. Additional signs include steep 'y' descent, hepatomegaly and rarely paradoxical pulse [31].**

**Presence of S3 or S4 may aid in assessing the severity. High grade AV blocks and bradyarrythmias are common [32,33]. Pericardial friction rub may be heard because RVI is usually transmural [31].**

RVI may be complicated by mechanical complications such as ventricular septal rupture [34,35], papillary muscle dysfunction giving rise to systolic murmur.

# Diagnosis

Unexplained hypotension or diminished cardiac output, or marked hypotension in response to small dose of nitroglycerine in patients with IWMI, should lead to the prompt consideration of the diagnosis. Echocardiography and gated blood pool scintigraphy are effective in diagnosing clinically significant RVI [36].

# Investigations

1.Electrocardiogram

**Lead $V_4R$ emerged as a reliable marker of site of coronary artery occlusion in acute inferoposterior myocardial infarction.**

**In patients with right ventricular infarction ECG may demonstrate 1mm of doming ST elevation in the right sided precordial leads, particularly in $V_4R$- $V_6R$. Right sided ST elevation, particularly in $V_4R$ is indicative of acute RVI [37,38,14] and correlates closely with occlusion of proximal RCA[38-,40].**

**An ST segment elevation in lead $V_4$ rather than in leads $V_1$- $V_3$ offers the highest specificity and accuracy in diagnosis [41].Lew and associates [42] found that ST segment depression in lead $V_2$ which is 50% or less the magnitude of ST segment elevation in lead aVF indicate right ventricular ischaemic injury.**

**Greater ST segment elevation in lead III than in lead II is also suggestive of RVI .In one study this finding had a sensitivity, specificity and positive and negative predictive value of 97%, 56%, 69% and 95% respectively [43].**

**Occasionally RVI may be associated with ST segment elevation in lead $V_1$ as well as other precordial leads (leads $V_2$ –$V_5$). This may mimic an AWMI [41,42,44] , however magnitude of ST segment elevation decreases from $V_1$-$V_5$ in RVI. ST segment elevation being maximal in lead $V_1$[22].**

**ST elevation in V4R is transient, resolving approximately in one half of patients in an older study [45] and presumably more frequently in the reperfusion era. Other causes of ST segment elevation**

**in the right-sided precordial leads include pulmonary embolism, pericarditis, and anteroseptal MI [27].**

## 2.Echocardiography

**Echocardiography is often an useful test that can be performed at the**

**bedside [,27,46,47] when the diagnosis of RVI is suspected. Right ventricular size and function and the degree of tricuspid insufficiency can all be evaluated. Contrast echocardiography can also detect a right to left shunt through patent foramen ovale[48]. The 2007 ACC/AHA [37,49] guidelines recommended the use of echocardiography in the evaluation of IWMI. Echocardiographic manifestations of RVI are abnormal RV free wall motion and RV dilatation [48], decreased descent of RV base, plethora of IVC.**

## Hemodynamic measurements

**Hemodynamic criteria for dominant RVI are**

**1.Right atrial pressure >10mm of Hg or a <5mm of Hg difference from mean pulmonary capillary wedge pressure [50,51] .**

**2.Right atrial pressure to pulmonary capillary wedge pressure ratio >0.8 [50].**

**3.Non compliant right atrial pressure wave pattern, prominent 'y' descent [50-53,31] .**

**4.Early diastolic drop and plateau in the right ventricular pressure tracing.**

**5.Normal right ventricular and pulmonary artery systolic pressures.**

**6.Increased ratio of RV to LV filling pressure.**

**7.Decreased and delayed RV function curve [54] .**

## 3.Radionuclide imaging

**Radionuclide ventriculography and technetium pyrophosphate scanning have acceptable sensitivities and specificities for making a diagnosis of RVI [14,27,55].**

**Radionuclide ventriculography can detect motion wall abnormalities and hypoperfusion in the affected right ventricle. It can also be used to quantitate both LV and RV ejection fraction.**

**Technetium scanning is particularly useful for late diagnosis since it shows areas of necrotic dying myocardium. Contrast enhanced**

**cardiovascular magnetic resonance was sensitive for detection of right ventricular involvement [56].**

## 4.Cardiac enzymes

**Increased magnitude of enzymes relative to the degree of left ventricular dysfunction is seen in RVI. Plasma BNP leval or higher in IWMI with RVI than isolated IWMI. Right ventricular involvement may be suspected when BNP levels are higher than 46 picogram [57] per ml.**

## 5.Cardiac catheterisation

**1. Involvement of right (usually) coronary artery or left circumflex coronary**

**artery.**

**2.Right ventricular akinesis.**

# Differential diagnosis

1.Cardiac tamponade

**This is distinguished by the characteristic pulses paradoxus, prominent 'x' descent of JVP, ECG features (low voltage complexes, electrical alterans) and echocardiography (by the presence of pericardial effusion and small RV).**

2.Pulmonary embolism

**The presentation of pulmonary embolism and RVI may share some features like hypoxia, clear lung fields, hypotension, shock, rise in serum troponin and right ventricular dysfunction in echocardiogram.**

**Nature of chest pain, electrocardiogram can be useful in making diagnosis. In pulmonary embolism dyspnoea is more severe and right ventricular and pulmonary pressures are higher than in RVI.**

3.Constrictive pericarditis

**Patient do not have chest pain or ECG changes. Echo shows a thickened pericardium with effusion.**

4.Tension pneumothorax

**In these patients dysnoea will be more severe and mediastinal shift**

**will be present.**

5. Restrictive cardiomyopathy

**Patient usually do not have acute chest pain. Echo is helpful (RV size**

**normal with increased myocardial thickness) in diagnosis.**

# Complications

1. **Cardiogenic shock.**
2. **Atrioventricular blocks.**
3. **Atrial tachyarrhythmias.**
4. **Tricuspid regurgitation [58].**
5. **Right atrial to left atrial shunting through patent foramen ovale or**

**ASD [59] can result in hypoxia in patient with RV failure .**

6. **RV thrombus.**
7. **Pulmonory embolism**
8. **Ventricular septal rupture [34,35] .**
9. **RV free wall rupture and tamponade.**
10. **Pericarditis.**

# Treatment

## 1. Fluid resuscitation

**Intravenous fluid ( usually isotonic saline ) should be given to patients with evidence of low cardiac output who have no pulmonary edema and low JVP [37].This is done to enhance left sided filling pressure by raising the central filling pressure in an attempt to maximize forward flow out of the RV [34,60,51] . In most cases several litres of saline infused rapidly until there is increase in pulmonary capillary wedge pressure to 15 mm of Hg**

**If central hemodynamic monitoring not available, one to two litres of**

**saline can be infused by closely maintaining BP, urine output and examining**

**patients for signs of pulmonary congestion.**

**Patients who are at risk of pulmonary edema should not receive rapid**

**volume administration.These patients include those with**

**1.prior myocardial infarction**

**2.acute, extensive LV infarction**

**3.associated mitral regurgitation**

**4.acute anterior myocardial infarction.**

## 2.Avoid drugs that decrease preload

**Nitrates reduce preload , while beta blocker and calcium channel**

**blocker can reduce heart rate and contractility and slow AV conduction.**

**Thus these drugs should be avoided in patients who are hemodynamically**

**unstable but can be tried with careful monitoring in those who are stable**

**and have a clear indication [34].**

## 3.Inotropic drugs

**When fluid resuscitation is insufficient inotropic and chronotropic**

**stimulation with dobutamine may increase forward flow and augment**

**cardiac output [37,61,62]. Dobutamine also act by reducing pulmonary vascular**

**resistance and therefore RV afterload.**

## 4.Pacing

Bradyarrhythmias can significantly worsen hemodynamic status. Atropine and aminophylline , if the patient does not respond, may be beneficial [63] but RV or AV sequential pacing may be necessary [34,64]. According to 2007 ACC/AHA guidelines, AV synchrony should be achieved and bradycardia should be corrected in all patients with RVMI [37,49].

## 5.Reperfusion

**Early reperfusion using either primary PCI or thrombolytic therapy**

**can preserve right and left ventricular function and reduce mortality and**

**morbidity [13,65,66]. In an analysis of benefits of thrombolysis in IWMI, the**

**mortality benefits was limited to the patients with RV involvement, with the**

**reduction in mortality well from 42-10% [67]. Patient without RVI had a similar mortality rate (6%-7%) with and without thrombolytic therapy.**

**Similarly in TIMI II trial thrombolytic therapy reduce incidence of RVI from 42% to 12% for patients with IWMI [65] . Radionuclide studies have confirmed a marked reduction in the extent of RVI in patients who achieved early reperfusion [68].**

Stepwise management of RV infarction

| | |
|---|---|
| **Recognition** | **ST segment elevation in lead II, III and aVF**<br>**plus ST segment elevation in leads $V_4R$ –$V_6R$** |

| | |
|---|---|
| **Reperfusion** | **Thrombolysis -Streptokinase, 1.5 MU given IV over 60 minutes (or) rt-PA (recombinant alteplase) given first in 15 mg IV bolus, then 50 mg given over 30 min followed by 35 mg given over 60 min (or) rt-PA(recombinant reteplase) given in 10-MU IV bolus, with 10-MU bolus 30 minutes later.**<br>**Angioplasty ,Coronary bypass surgery** |
| **Volume loading** | **Normal saline, 40 ml per minute given IV upto**<br>**total of 2litres, keeping RA pressure at lessthan**<br>**18 mm Hg, hemodynamic monitoring required.** |
| **Inotropic support** | **Dobutamine 2 to 5 microgram per Kg per minute given IV, with dose increased every 5 to 10 minutes upto 15 to 20 microgram per Kg** |

| | |
|---|---|
| | **per minute.** |
| **Rate and Rhythm control** | **Symptomatic bradycardia-atropine, 0.521 mg given IV every five minutes upto total of 2.5 mg. AV block-AV sequential pacing (usually short term).** |
| **Complications** | **LV ischaemic dysfunction-Judicious afterload reduction(managed with ACE inhibitor); Volume restriction. Cardiogenic shock-aortic balloon pump. Interventricular septal rupture-emergency surgical repair. RV capillary muscle rupture and tricuspid regurgitation-emergency surgical repair [58].** |

**MU=Mega Unit; IV=Intravenous; rt-PA=recombinant tissue Plasminogen**

**Activator; RA=Right Atrial; AV=Atrio Ventricular; LV=Left Ventricular;**

**RV=Right Ventricular.**

**Successive steps usually implies lack of success with preceding steps or increased severity of the patient's condition. The preeminent first**

**step after recognition is reperfusion. Angioplasty resumes failed thrombolysis, Coronary bypass surgery resumes failed angioplasty or the presence of multivessel disease. Successful reperfusion returns RV function and prevents the need for volume loading. When volume loading fails, inotropic support may be required. When atropine fails to correct symptomatic bradycardia or relieve AV block, pacing may be needed. The complications and their strong counter measures resume severe, unrelieved RV ischaemic damage.**

# Prognosis

**Presence of RVI adversely affects early outcome after an MI, and persistent RV dysfunction adversely affects late prognosis.**

## Early prognosis

**Individual studies and metaanalysis have shown that RV involvement in patients with an acute IWMI is associated with a worse in hospital**

**outcome primarily due to persistent hypotension [13,14,35,36] .**

## Long term prognosis

**The prognosis of patients who survive an acute RVI is primarily determined by the extent of LV involvement.**

# Materials and methods

## Setting

**A Cohort study and analysis of patients admitted with inferior wall myocardial infarction in Intensive Care Unit, Government Royapettah Hospital.**

## Study population

**Study was conducted over a period of 6 months Sep2007-Feb2008. 50 patients admitted in Intensive Care Unit, Government Royapettah Hospital were chosen for study and followed up for entire period of stay in hospital.**

## Inclusion criteria

1. **Patients admitted with chest pain which had began less than 24 hours before admission.**
2. **ST segment elevation of 1mm or more in two or more of the leads LII, LIII and aVF.**

## Exclusion criteria

1. **Previous history of anterior wall infarction.**

2. **Associated anterior wall infarction.**

**Information collected consists of basic data including name, age, sex,**

**presenting complaints, history of diabetes , systemic hypertension, chronic**

**respiratory illness/bronchial asthma. Vital signs were noted. Jugular venous**

**pressure was noted at 45 degree supine position and if found to be more**

**than 8cm, was taken as elevated.**

**Detailed systemic examination done to look for signs of cardiac**

**failure. Signs of left ventricular failure evidenced by bibasilar rales and**

**LVS3/LVS4, if present were noted.**

**12 lead ECG with right precordial leads was taken at admission and**

**at regular intervals thereafter and its findings noted. ST segment elevation >**

**1mm in any of right precordial leads was taken as RVI. Amount of ST**

**elevation and QRS morphology in lead $V_4R$ was noted. Posterior wall**

**myocardial infarction was noted if ST segment elevated in posteriorly**

**placed leads $V_7$,$V_8$ and $V_9$. Also lateral extension of myocardial infarction**

**was noted.**

**Blood sugar, serum cholesterol, SGOT levels were taken. Echocardiography done prior to discharge.**

# Observations and analysis

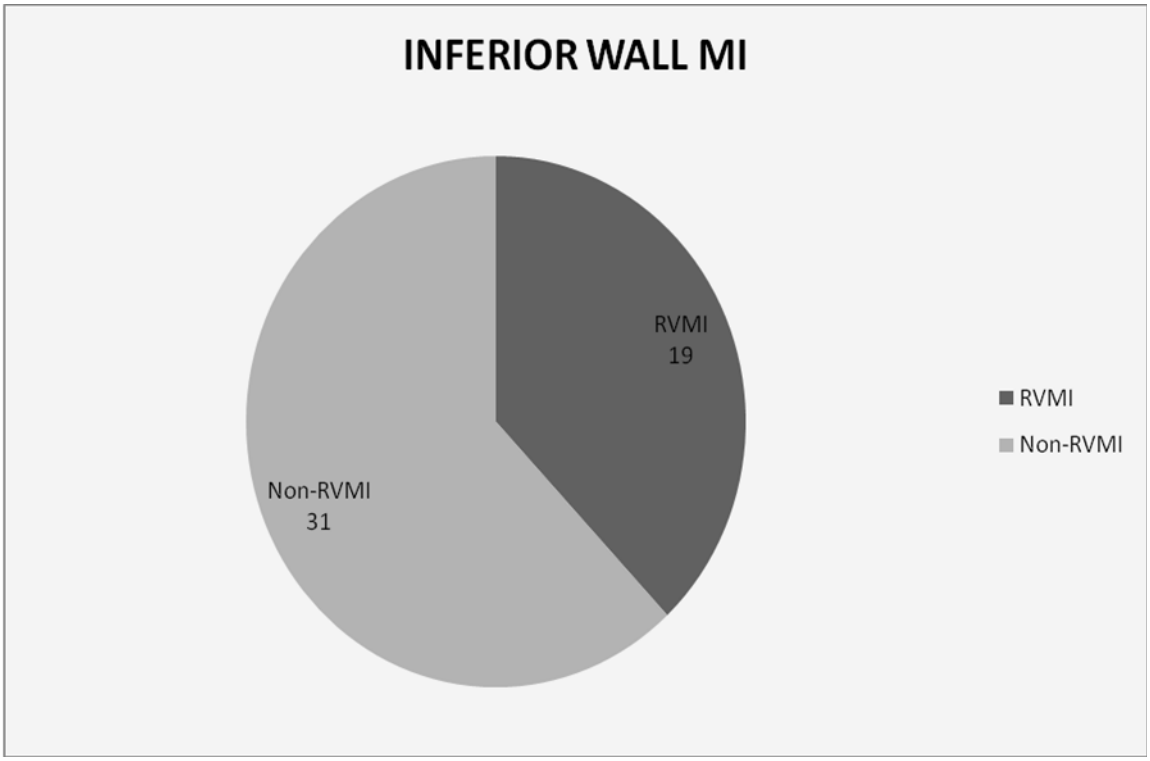
## **1.** Incidence of RVI

**Total number of IWMI patients = 50**

**with RVI = 19**

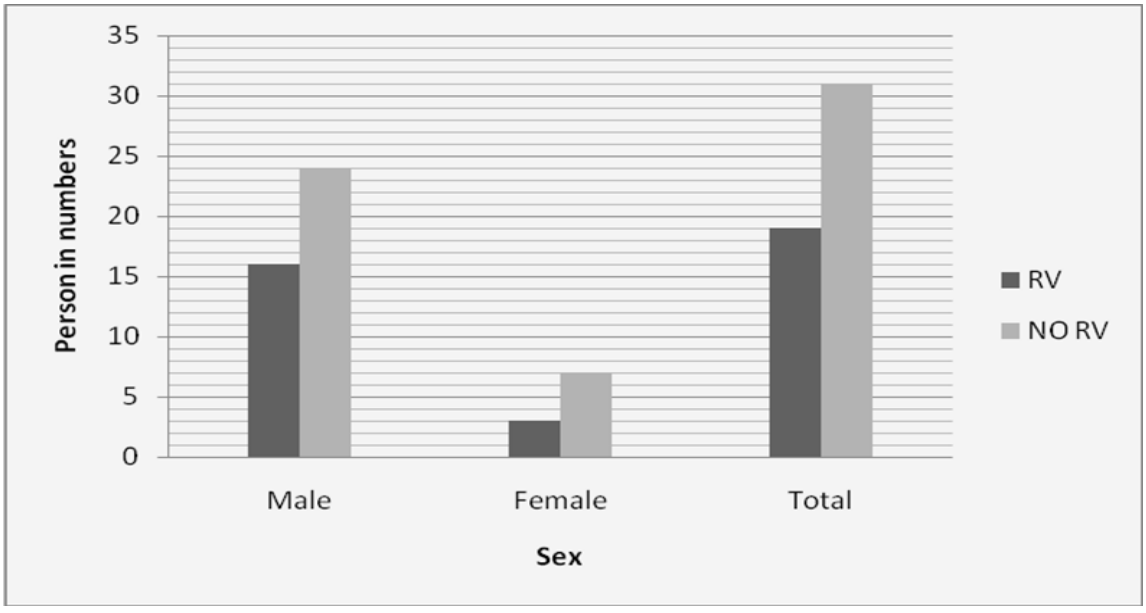
**without RVI = 31**

**Incidence of RVI in IWMI was found to be 38%.**



**2.** Sex distribution

| Sex | IWMI | |
|---|---|---|
| | **With RVI** | **Without RVI** |
| **Males** | **16** | **24** |
| **Females** | **3** | **7** |



**There were 40 males and 10 females in this study. Of 40 males, 16 had RVI and 24 did not have RVI. Of the 10 female, 3 had RVI and 7 did not have RVI.**

**Male to Female ratio.**

**Total IWMI = 4:1**

**With RVI = 5:1**

**Without RVI = 3:1**

## 3. Age group

| IWMI | Range(Years) | Mean(years) | Standard deviation |
|---|---|---|---|
| With RVI | 38-74 | 52.2 | 9.103 |
| Without RVI | 38-75 | 54.5 | 10.14 |

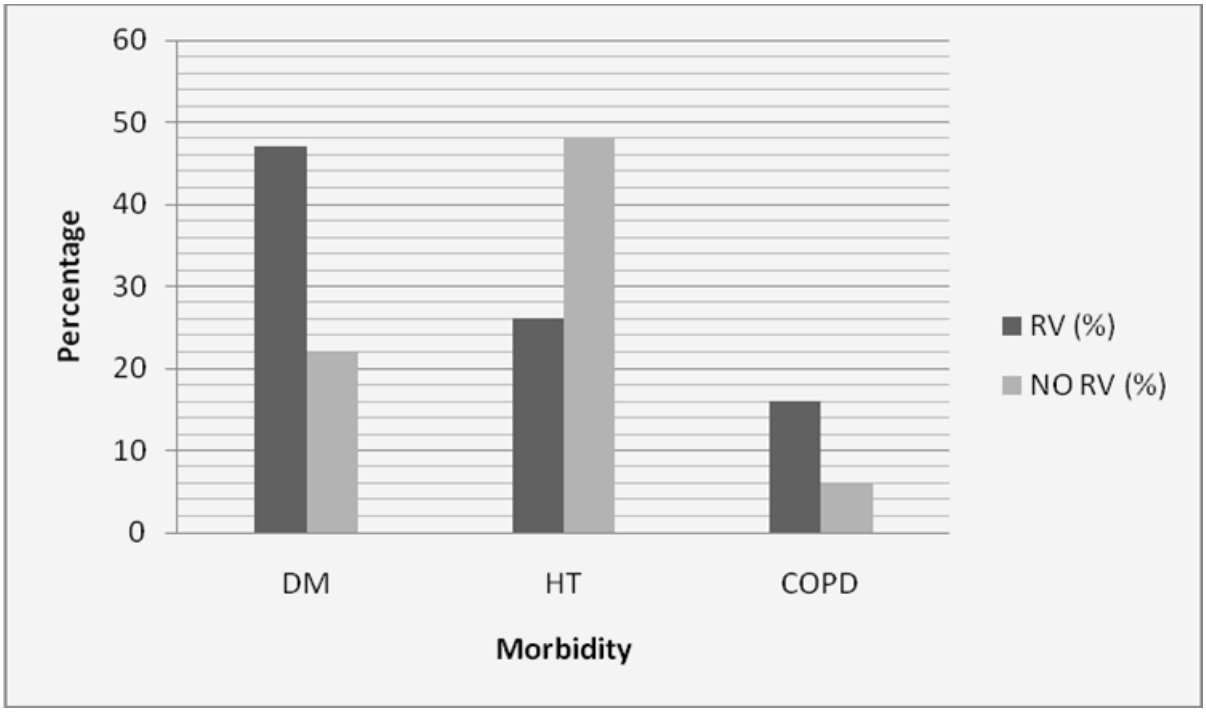
**Average age of IWMI patients with RVI was 52.2 with a standard deviation**

**of 9.103 and in IWMI patients without RVI it was 54.5 with a standard**

**deviation of 10.14.**

## 4. Clinical history

| Clinical history | With RVI | | Without RVI | |
|---|---|---|---|---|
| | No | % | No | % |
| 1.H/O DM | 5 | 26% | 15 | 48% |
| 2.H/O HT | 9 | 47% | 7 | 22% |
| 3.H/O Chr. Resp. illness | 3 | 16% | 2 | 6% |



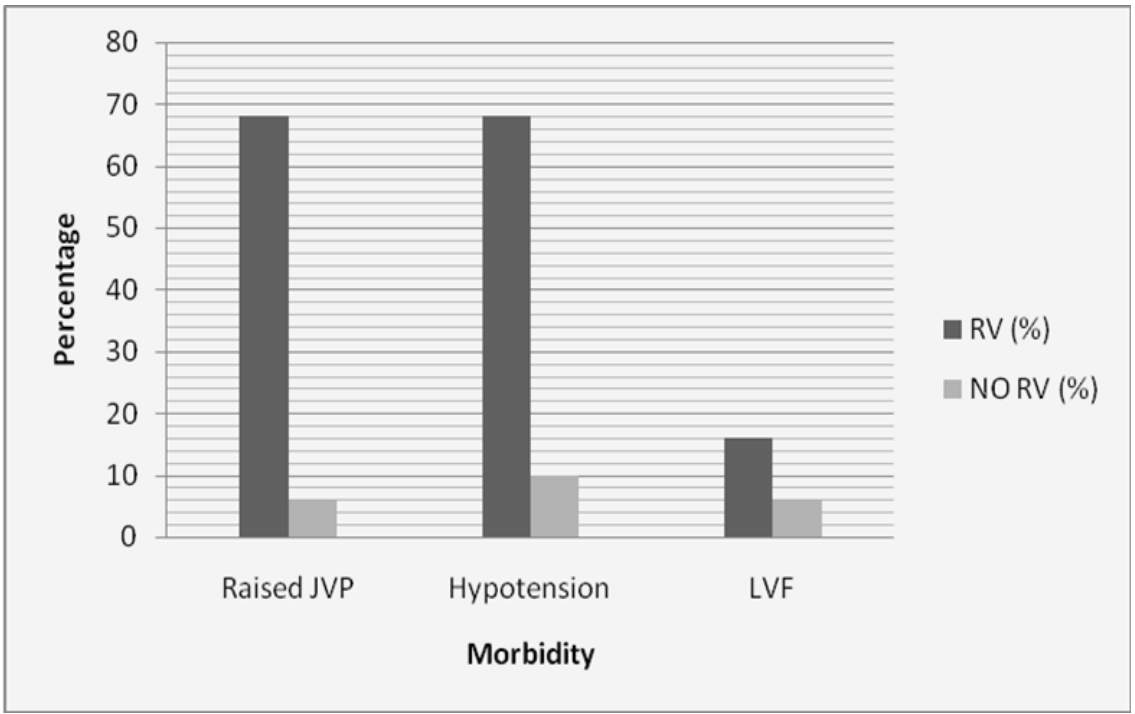

**26% of RVI patients had diabetes(5), compared to 48% without RVI(15).**

**47% of RVI patients had hypertension(9), compared to 22% without RVI(7).**

**16% of RVI patients had a history of chronic respiratory illness(3), compared to 6% without RVI(2)**

## 5. Clinical manifestations

| Clinical | With RVI(19 ) | | Without RVI(31) | |
|---|---|---|---|---|
| | No | % | No | % |
| 1.Hypotension | 13 | 68% | 3 | 10% |
| 2.JVP elevation | 13 | 68% | 2 | 6% |
| 3.LVF | 3 | 16% | 2 | 6% |



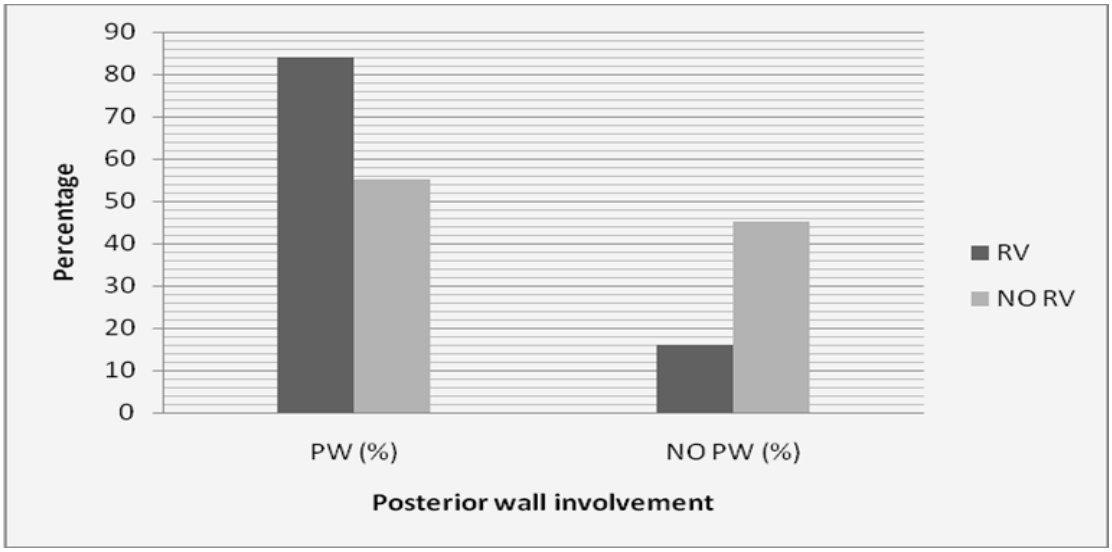
**Hypotension was present in 68% of patients with RVI(13) and 10% of patients without RVI(3).**

**JVP was elevated in 68% of patients with RVI(13) and 6% of patients without RVI(2).**

**Left ventricular failure was present in 16% of patients with RVI(3) and 6% of patients without RVI(2).**

**6.** Posterior wall myocardial infarction

| Inferior wall MI | Total | Posterior wall MI | | | | P value |
|---|---|---|---|---|---|---|
| | | Present | | Absent | | |
| | | No | % | No | % | |
| With RVI | 19 | 16 | 84 | 3 | 16 | 0.06* |
| Without RVI | 31 | 17 | 55 | 14 | 45 | |



**Of the 19 patients with RVI, 84% also had PWMI (16) and of the 31 patients**

**without RVI, 55% had PWMI(17).**

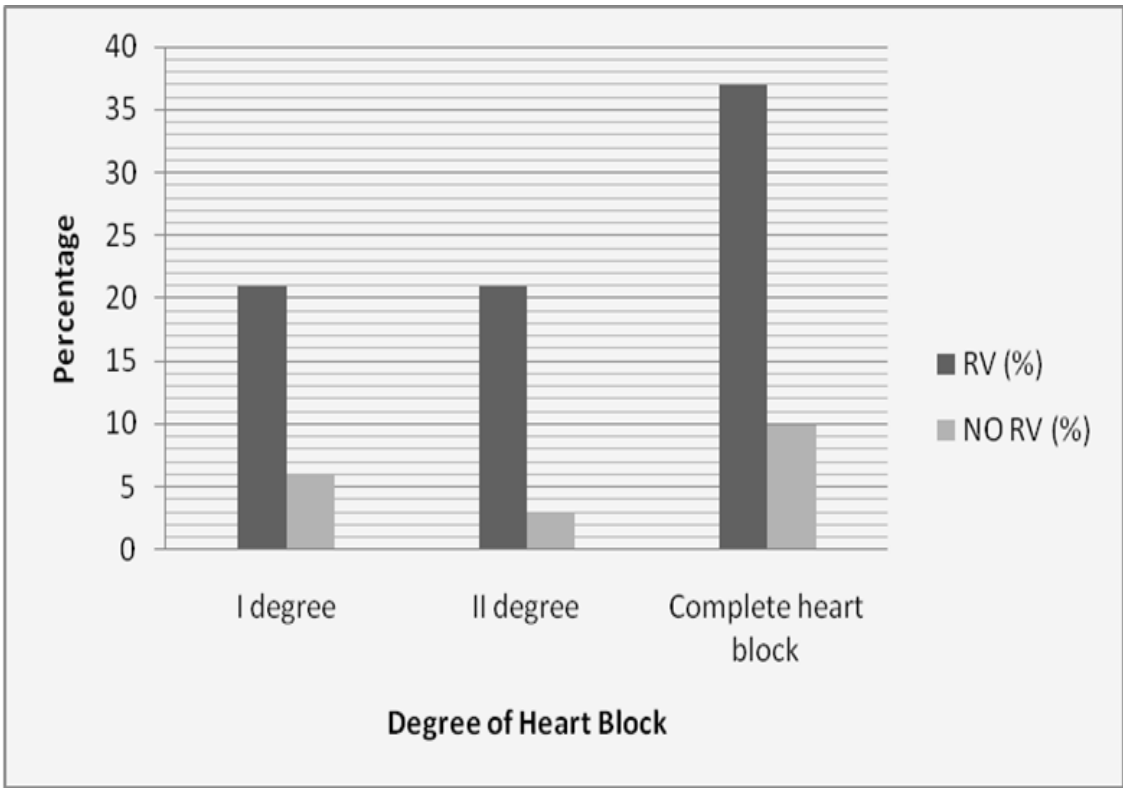
**So, the association of RVI with PWMI, in the setting of acute IWMI was**

**found to be statistically significant.**

**7.** Clinical course and complications

| Clinical course & complications | With RVI(19%) | | Without RVI(19%) | |
|---|---|---|---|---|
| | No | % | No | % |
| 1.Sinus bradycardia | 4 | 21% | 4 | 13% |
| 2.I$^{o}$ AVB | 4 | 21% | 2 | 6% |
| 3.II$^{o}$ AVB | 4 | 21% | 1 | 3% |
| 4.CHB | 7 | 37% | 3 | 10% |
| 5.VPD | 2 | 10% | 2 | 6% |
| 6.Sinus arrest | 2 | 10% | 0 | - |

| | | | | |
|---|---|---|---|---|
| **7.VT** | **1** | **5%** | **1** | **3%** |




**Sinus bradycardia was seen in 4 patients (21%) with RVI and in 4 patients**

**(13%) without RVI.**

**I degree atrioventricular block was seen in 4 patients (21%) with RVI and**

**in 2 patients (6%) without RVI.**

**II degree artrioventricular block was seen in 4 (21%) patients with RVI and**

**in 1 (3%) patient without RVI.**

**Complete heart block was seen in 7 patients (37%) with RVI and in 3**

**patients (10%) without RVI.**

**Frequent VPD noted in 2 patients (10%) with RVI and in 2 patients (6%)**

**without RVI.**

**Sinus arrest was noted in 2 patients (10%) with RVI, not in patients without**

**RVI.**

**Ventricular tachycardia was noted in 1 patient (5%) with RVI and in 1**

**patient (3%) without RVI.**

# 8. Comparison of data in patients with and without RVI

| Data | Total | WithRV | | Without RV | | P value |
|---|---|---|---|---|---|---|
| | | Num | % | Num | % | |
| 1.DM | 20 | 5 | 25% | 15 | 75% | 0.1 |
| 2.HT | 16 | 9 | 56% | 7 | 44% | 0.1 |
| 3.Chr resp. illness | 5 | 3 | 60% | 2 | 40% | 0.3 |
| 4.Hypotension | 16 | 13 | 81% | 3 | 19% | 0.00002* |
| 5.JVP | 15 | 13 | 87% | 2 | 13% | 0.00005* |
| 6.LVF | 5 | 3 | 60% | 2 | 40% | 0.3 |
| 7.I $^{0}$ AVB | 6 | 4 | 67% | 2 | 33% | 0.1 |
| 8.II$^{0}$ AVB | 5 | 4 | 80% | 1 | 20% | 0.05* |
| 9.CHB | 10 | 7 | 70% | 3 | 30% | 0.02* |
| 10.VPD | 2 | 1 | 50% | 1 | 50% | 0.3 |

**20 patients of IWMI had diabetes mellitus of whom 5(25%) had RVI and**

**15(75%) had no RVI. 16 patients had hypertension of whom 9(56%)**

**had RVI and 7(44%) had no RVI. 5 patients had history of chronic respiratory illness of whom 3(40%) had RVI and 2(60%) had no RVI.**

**Thus the risk factors of diabetes mellitus hypertension and chronic respiratory illness were not found to be statistically increased in patients who had RVI.**

**16 patients had hypotension of whom 13(81%) had RVI and 3(19%) did not have RVI. 15 patients had elevated JVP of whom 13(87%) had RVI and 2(13%) did not have RVI. 5 patients had left ventricular failure of whom 3(60%) had RVI and 2(40%) did not have RVI.**

**Hypotension and elevated JVP were found to be present in statistical significance in patients with RVI than in those without RVI, whereas left ventricular failure was not found to be increased to a significant extent in RVI.**

**I degree atrioventricular block present in 6 patients of whom 4(67%) had**

**RVI and 2(33%) did not have RVI. II degree atrioventricular block present**

**in 5 patients of whom 4(80%) had RVI and 1( 20%) did not have RVI. Complete heart block was noted in 10 patients of whom 7 had RVI(70%)**

**and 3(30%) did not have RVI.**

**II degree atrioventricular block and complete heart block were found**

**to be significantly increased in IWMI patients with RVI than in those**

**without RVI.**

9.Investigation

1.Cardiac enzymes

| **Inferior wall myocardial infarction** | **SGOT** | |
|---|---|---|
| | **Mean** | **Std dev** |
| **1.With RV** | **136.15** | **92.34** |
| **2.Without RV** | **80.94** | **62.53** |

**P value 0.03***

**Patients with RVI had mean SGOT level of 136.15 and patients without**

**RVI had a mean level of 80.94.**

**Thus patients with RVI had a significantly higher SGOT level than**

**patients without RVI.**

2.ECG- Right precordial leads

**(i) Relation between magnitude of ST segment elevation in lead $V_4R$ and clinical picture.**

| ST elevation | 1 mm | 2 mm | 3 mm |
|---|---|---|---|
| Total | 6 | 8 | 5 |
| 1.Death | 0 | 1 | 1 |
| 2.Hypotension | 2 | 7 | 4 |
| 3.JVP ↑ | 2 | 7 | 4 |
| 4.LVF | 0 | 1 | 2 |
| 5.I $^0$ | 2 | 2 | 0 |
| 6.II $^0$ | 0 | 2 | 2 |
| 7.III $^0$ | 1 | 3 | 3 |
| 8.VT | 0 | 0 | 1 |

**Majority of patients had ST segment elevation more than 1mm. Both the patients who expired had ST segment elevation of more than 1mm.**

**(i) QRS morphology in lead $V_4R$**

| Morphology | With RVI | |
|---|---|---|
| | No | % |
| QS | 14 | 74% |
| rS | 3 | 16% |
| Rs | 1 | 5% |
| rsR’ | 1 | 5% |

| Morphology | Without RVI | |
|---|---|---|
| | No | % |
| QS | 8 | 26% |
| rS | 17 | 55% |
| R | 2 | 6% |
| Rs | 4 | 13% |

**74% of patients with RVI had QS morphology in lead $V_4R$, whereas only**

**26% of patients without RVI had the same. 16% of patients with RVI had**

**rS morphology whereas 55% of patients without RVI had rS morphology**

**in lead $V_4R$.**

**(i) Duration of ST segment elevation in lead $V_4R$.**

| **ST elevation after** | **No** | **%** |
|---|---|---|
| **12 hrs** | **7** | **37%** |
| **24hrs** | **2** | **10%** |
| **48hrs** | **-** | **-** |

**By 12 hours ST segment elevation disappeared in 63% of patients of RVI**

**and by 48 hours disappeared in all patients.**

3.Echocardiography

**Echo done in all 19 patients showed inferior wall hypokinesia. Echo did not show right ventricular hypokinesia. There was no evidence of right ventricular thrombus.**

10 .Treatment

**a.Thrombolysis**

| **Thrombolysis** | **With RVI** | **Without RVI** |
|---|---|---|
| **Done** | **14** | **21** |
| **Not done** | **5** | **10** |

**35 patients in total were thrombolysed of whom 14 had RVI and 21 did not have RVI.**

**b.Other modalities**

| | | |
|---|---|---|
| **Thrombolysis** | **14** | **74%** |
| **Fluids** | **11** | **58%** |
| **Inotropes** | **6** | **32%** |
| **Atropine** | **4** | **21%** |

**Volume expansion was done in 11 patients(58%) of RVI and inotropic support was given to 6 patients(32%). Atropine was given in 4**

**patients(21%).**

1. Outcome

| Outcome | Total | With RVI | Without RVI | P value |
|---|---|---|---|---|
| **Mortality** | **2** | **2** | **-** | **0.05** |

**Both the patients who expired in the study had RVI. Thus in hospital**

**mortality was found to be significantly increased in patients with RVI. Both**

**the patients were thrombolysed and given fluids and inotropic support, the**

**primary cause of death being cardiogenic shock.**

# Discussion

**1.** Incidence

**Right ventricular infarction is associated with infarction of the**

**inferior wall of left ventricle, occurring in more than 1/3 of such cases and usually the posterior wall was also involved [23,34,69]. Depending on the diagnostic criteria used, the incidence of right ventricular infarction in patients with inferior myocardial infarction ranges from 10%-15%. In this study also, right ventricular infarction was found to be significantly associated with posterior wall infarction in the setting of acute inferior wall myocardial infarction.**

**Incidence from other study:**

**Wackers et al [55] -37.5%**

**(Based on radionuclide scnitigraphy)**

**Braat et al [45] -43%**

**(Based on ECG)**

**Dell Italia et al [51] -28%**

**(Based on hemodynamic measurements)**

**Zehender et al [12] -54%**

**Gumina et al- -18%**

**In our study, based on right precordial ECG the incidence of right ventricular infarction was 38% among patients with inferior wall infarction.**

## 2. Age and Sex wise Analysis

**Mean age in patients with right ventricular infarction being 52.2 and without right ventricular infarction being 54.5. No significant difference in age was noted. Similar finding was noted in male to female ratio.**

**Patients with right ventricular infarction - 5:1.**

**Patients without right ventricular infarction - 3:1.**

## 3. Clinical History:

**Peter et al [70] studied a model of myocardial infarction and found that experimentally induced right ventricular hypertrophy rendered right ventricle susceptible to infarction and impaired collateralization of occluded right coronary artery in some cases.**

**Ratliff and Hackel [71] observed that isolated right ventricular infarction**

**are rarely seen at autopsy except in cases of right ventricular hypertrophy**

**associated with pulmonary hypertension.**

**In this study, no significant increase in incidence of diabetes**

**mellitus, hypertension, chronic obstructive pulmonary diseases were**

**noted in right ventricular infarction patients.**

**4.** Clinical presentation:

**Mehta et al [7], found that right ventricular infarction occurred in**

**nearly half of patients with inferior wall infarction and was associated**

**with the three fold increase in combined incidence of early mortality,**

**cardiogenic shock, ventricular tachyarrhythmia and advanced heart block**

**as compared to inferior wall myocardial infarction alone.**

**Erhardt et al [12] observed right heart failure in 76% with right**

**ventricular infarction and 27% without right ventricular infarction .Left**

**heart failure noted in 98% and hypotension in 65% of cases with right**

**ventricular infarction, 27% and 25% respectively in patients without right**

**ventricular infarction.**

**Cintron et al [30] and Cohn[5] described a high incidence of systemic venous congestion and hypotension in their patients with right ventricular infarction.**

**HIP-2 trial showed that, 1/3 of inferior wall myocardial infarction patients had right ventricular involvement [23,34]. These patients had higher 30 day mortality when compared to those without right ventricular infarction.**

**In this study, incidence of hypotension and JVP is 68% in patients with right ventricular infarction, significantly higher than in patients without right ventricular infarction, which is 10% and 6% respectively. Presence of left ventricular failure is 16% in patients with right ventricular infarction and 6% in patients without right ventricular infarction.**

5. Conduction disturbance:

**Braat et al[39] reported the incidence of sinus bradycardia with**

**inferior wall myocardial infarction as 10%-30% and conduction block as**

**5% to 25%. In case of right ventricular infarction, atrioventricular nodal**

**conduction disturbance occurred in 48% of cases and atrioventricular**

**dissociation was noticed in 30% to 40% of cases.**

**Cohn [5] et al found complete heart block in 40% of patients**

**with right ventricular infarction.**

**In Zehender study[14], sinus bradycardia was found in 9% of**

**patients with right ventricular infarction and in 3% of patients**

**without right ventricular infarction, complete heart block in 16% of**

**patients with right ventricular infarction and in 4% of patients**

**without right ventricular infarction.**

**In this study, sinus bradycardia, $I^{O}$ AV block , $II^{O}$ AV block**

**and complete heart block were found in 21%, 21%, 21% and 37%**

**respectively in patients with right ventricular infarction and 13%, 6%, 3%**

**and 10% respectively in patients without right ventricular infarction . $II^{O}$**

**AV and complete heart block were significantly higher in patients with**

**right ventricular infarction than in those without right ventricular infarction.**

**Values are similar to those obtained by Braat et al but higher than those of Zehender et al. In our study, there is no significant difference in ventricular tachycardia incidence was noted.**

## **6.** Investigation

### **1.** Cardiac enzymes**:**

**Erhardt et al has reported significantly higher levels of SGOT, SGPT and LDH in patients with right ventricular infarction suggesting more extensive myocardial damage.**

**In this study, mean SGOT level in patients with right ventricular infarction was found to be significantly higher than the mean SGOT level in those without right ventricular infarction.**

### **2.** Right precordial ECG changes**:**

**Various study by Erhardt et a [12], Zeymer et al[13], and Andersan et al**

**have shown that a true right ventricular lead (lead $V_4R$) was of value in**

**the diagnosis of right ventricular infarction in patients with acute inferior**

**wall myocardial infarction.**

**This study showed majority of patients had ST elevation in all**

**right precordial leads. Magnitude of ST segment elevation decreased**

**from $V_3R$ to $V_6R$ in most cases.**

**Braat et al[45] reported that ST segment elevation in lead $V_4R$**

**resolved within 10 hours after chest pain in 50% of patients.**

**In this study, ST segment elevation resolved within 12 hours in**

**63% of patients. No direct relation between magnitude of ST segment**

**elevation in right precardial leads and the course of disease made out.**

Q wave in right ventricular infarction:

**In normal subjects, an rS pattern is always present in lead $V_3R$**

**and usually in lead $V_4R$.**

**In this study, 74% of patients in right ventricular infarction showed**

**QS complex. This is similar to results of Morgera et al [72], who showed 78%**

**of right ventricular patients had a QS complex.**

**3.** Echocardiography

**All patients showed hypokinesia of diaphragmatic wall. Echo did not show**

**any right ventricular hypokinesia, because right ventricle will recover**

**earlier after infarction, say within 6 hours.**

**7.** Treatment

**74% of patients were thrombolysed. 58% of right ventricular infarction patients required fluids, 32% were given inotropic support and none required pacing.**

**8.** Outcome

**Similar to other studies, mortality was significantly higher in patients**

**who sustained a right ventricular infarction.**

# Summary and conclusion

**50 patients of acute inferior wall myocardial infarction were studied for the presence of right ventricular infarction by ECG, clinical features and immediate prognosis were compared with patients who showed no evidence of right ventricular infarction.**

- **The incidence of RVI was 38%.**
- **There was no significant difference in age or sex distribution.**
- **There was no significant difference in the risk factors, diabetes, hypertension and chronic respiratory illness between the two groups.**
- **Hypotension and elevated jugular venous pressure were significantly found in those who sustained RVI than in those who did not.**
- **II $^{0}$ AVB and CHB were found to be significantly increased in those with RVI.**

- **Other arrhythmias were not significantly affected by associated RVI.**

- **RVI were found to be significantly associated with posterior wall myocardial infarction in the setting of acute inferior wall myocardial infarction.**

- **This study also points out the utility of lead $V_4R$ in diagnosing RVI and confirms the specificity of QS waves in $V_4R$ in diagnosing RVI. The ST segment elevation in lead $V_4R$ was noted to be transient.**

- **Mortality in acute stages was found to be significantly affected by the presence of RVI in patients with IWMI.**